Otoconial Assessment, SVV & VEMP:
A Comprehensive Clinical Review

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Section 3C — Otolith & Otoconial Assessment | Vestibular Function Testing Series

How to Use This Review

This document is the companion clinical literature review to the otolith function, subjective visual vertical, and vestibular evoked myogenic potentials video series on the ADC education hub at www.australiandizzinessclinics.com. It is designed for vestibular physicians, audiologists, and neurologists building expertise in laboratory vestibular function testing.

The review follows clinical testing sequence: from theoretical foundations and neural substrates through methodology, normative values, interpretation frameworks, and clinical application. Callout boxes throughout identify clinically high-yield points and evidence-based pearls.

Callout box guide:

 Clinical Insight: *Clinically relevant observations derived directly from the basic science — the bridge between laboratory findings and patient management.*

 Clinical Pearl: *High-yield, memorable clinical points — the key facts that separate a competent clinician from an expert in vestibular function testing.*

 Key Point: *Foundational concepts and summary statements that anchor the clinical framework. Master these to interpret the full testing battery.*

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Otolith Organ Function in Vestibular Medicine

Utricle, Saccule, and Central Connections and its Assessment in Clinical Setting

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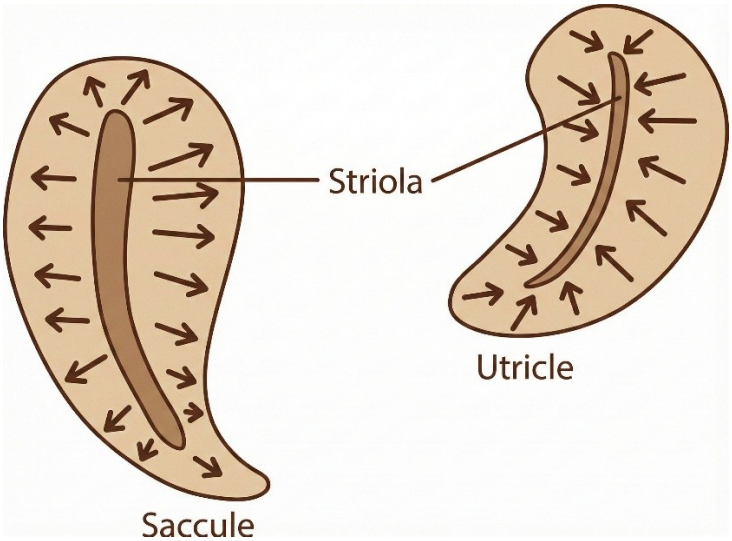
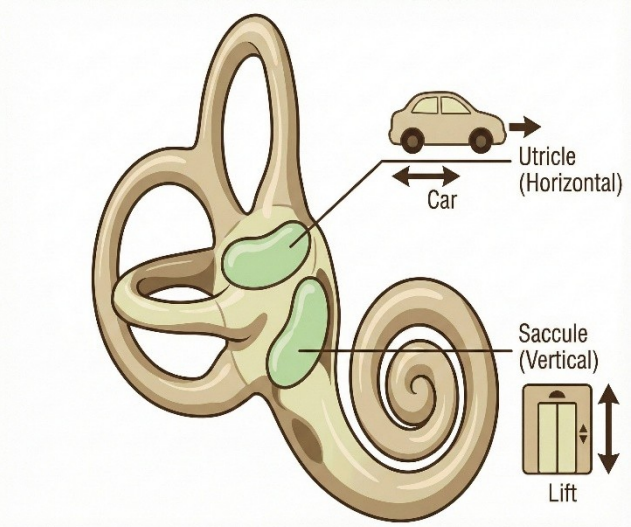
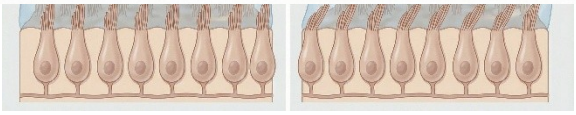
Introduction

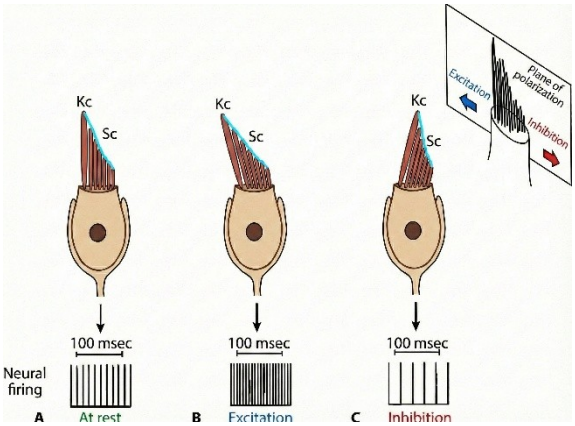
For the vestibular physician, the otolith organs (utricle and saccule) represent an often-underappreciated half of the inner ear's balance system. These gravity sensors provide a continuous reference for "which way is up," detecting linear accelerations and head tilt relative to gravity – functions complementary to the angular motion sensing of the semicircular canals [1]. In clinical practice, subtle signs arising from otolith dysfunction (e.g. abnormal perception of vertical or skewed eye alignment) can be as diagnostically decisive as canal-mediated vestibulo-ocular reflex (VOR) abnormalities. Mastery of otolithic anatomy, physiology, and test interpretation allows the vestibular physician to localize lesions more precisely and to distinguish peripheral otolith disorders from central graviceptive pathway lesions [2]. This review provides a comprehensive overview of utricle and saccule function and their central connections, beginning with foundational anatomy and physiology (including brief phylogenetic perspectives), then detailing the role of otolith organs in normal vestibular function. A major focus is devoted to clinical assessment of otolith function – both bedside and laboratory – with an in-depth examination of vestibular evoked myogenic potentials (VEMP) including indications, contraindications, techniques, parameters, limitations, and interpretive caveats. We also review other otolith-specific tests such as the subjective visual vertical (SVV) "bucket test," the ocular tilt reaction, and positional skew deviation testing.

Anatomy and Physiology of the Otolith Organs



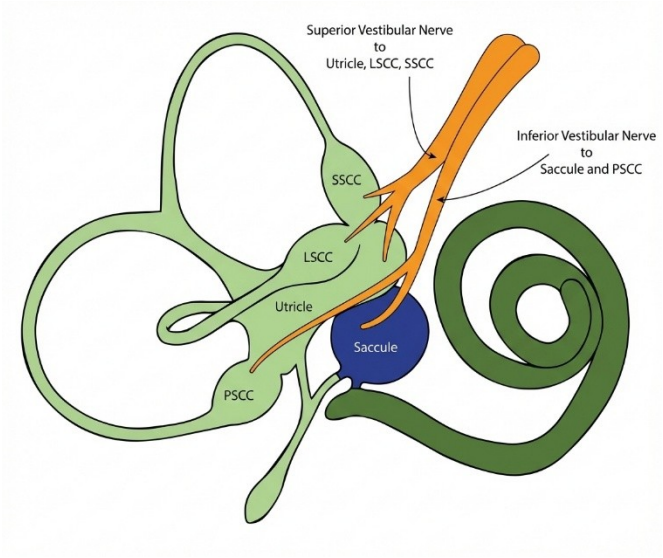


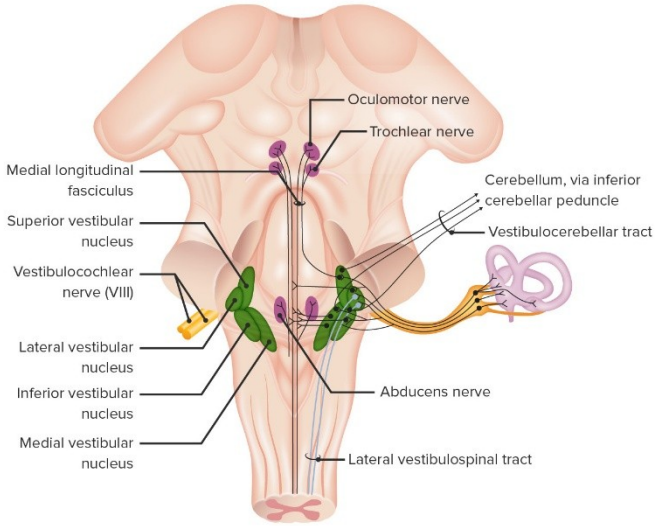




Macular Structure and Function: The utricle and saccule (the otolith organs) are sac-like inner ear organs containing specialized sensory epithelium called maculae. Each macula is a patch of hair cells topped by a gelatinous otolithic membrane embedded with calcium carbonate crystals (otoconia). The weight of the otoconia gives the otolithic membrane inertia. When the head tilts or translates linearly, gravitational or inertial forces cause the heavy otoconial membrane to deflect, bending the stereocilia of underlying hair cells. This mechanical shearing opens ion channels and modulates the hair cell's afferent firing rate: stereocilia bending toward the kinocilium depolarizes (excites) the cell, while bending away hyperpolarizes (inhibits) it. In contrast to the semicircular canals (which have hair cells aligned uniformly in each crista), the otolithic maculae have a complex polarization pattern. A curving central zone called the striola divides each macula into two regions with opposing hair cell orientation [3]. In the utricular macula, kinocilia are oriented toward the striola, whereas in the saccular macula they are oriented away from the striola. This arrangement means that any given linear motion will excite some hair cells while inhibiting others, allowing the utricle and saccule together to cover the full range of linear directions in the horizontal and vertical planes. The utricle's macula lies roughly in the horizontal plane (when the head is upright) and is most sensitive to horizontal translations and head tilts in the roll plane, while the saccular macula is oriented vertically (parasagittal plane) and responds to vertical motions (up-down translations) and pitch-plane head tilts [4]. Importantly, because gravity is a constant linear acceleration, the macular hair cells provide a tonic signal encoding head position relative to Earth's vertical. This allows the otolith organs to sense static head tilt as well as transient linear accelerations. Once a constant-velocity motion is reached (no net acceleration), the otolithic deflection subsides as the otoconial mass equilibrates, meaning the organs respond primarily to changes in linear velocity (i.e. acceleration) and sustained head tilts.

Neural Innervation and Central Pathways: The hair cells of the utricle and saccule synapse on bipolar neurons whose cell bodies reside in Scarpa's ganglion. Notably, the vestibular nerve has two functionally distinct divisions: the superior division carries afferents mostly from the utricle and the anterior and horizontal semicircular canals, while the inferior division carries afferents from the saccule and posterior canal [5]. This anatomy explains why isolated vestibular neuritis can sometimes affect only "half" the labyrinth (e.g. superior vestibular neuritis involving utricle and lateral/superior canals but sparing saccule). Upon entering the brainstem, the primary otolith afferents project to the four vestibular nuclei (medial, lateral [Deiters'], superior, and inferior nuclei in the pontomedullary junction) and also send collaterals to the cerebellum (especially the vestibulocerebellum) via the juxtarestiform body. Within the vestibular nuclear complex, otolithic signals are distributed in a somatotropic fashion: the lateral vestibular nucleus predominantly processes otolith inputs for spinal reflexes (balance and extensor tone), while the superior and medial vestibular nuclei integrate both canal and otolith inputs for ocular reflexes [2]. Crucial premotor pathways originate here. For example, neurons in the vestibular nuclei give rise to the vestibulo-ocular pathways ascending in the medial longitudinal fasciculus (MLF) to drive extraocular muscle nuclei (III, IV, VI). These include utriculo-ocular projections that mediate eye movements in response to head tilts or translations. Similarly, vestibulospinal pathways arise for postural control: the lateral vestibulospinal tract (LVST) descends ipsilaterally from Deiters' nucleus through the anterior spinal cord to facilitate extensor (antigravity) muscles for balance, while the medial vestibulospinal tract (MVST) descends mostly bilaterally from the medial vestibular nucleus via the MLF to cervical levels, coordinating neck muscles (including the sternocleidomastoid) for head stabilization. The MVST is the pathway underlying the vestibulo-collic reflex, by which otolithic stimulation can induce compensatory neck muscle responses. A clinically relevant point is that lesions of the MVST (for instance, in the brainstem) may abolish the ipsilateral cervical vestibular myogenic potential by disrupting this descending pathway – highlighting that an absent VEMP can sometimes reflect central pathway damage rather than peripheral end-organ loss.





Phylogenetic Perspective: The otolith organs are phylogenetically ancient. In evolutionary terms, gravity detection was such a fundamental requirement that even the simplest multicellular animals developed statocyst organs (containing mineralized statoliths) for orientation. The otolithic system predates the development of elaborate semicircular canals – for example, primitive vertebrates like lampreys possess only two semicircular canals but retain fully functional

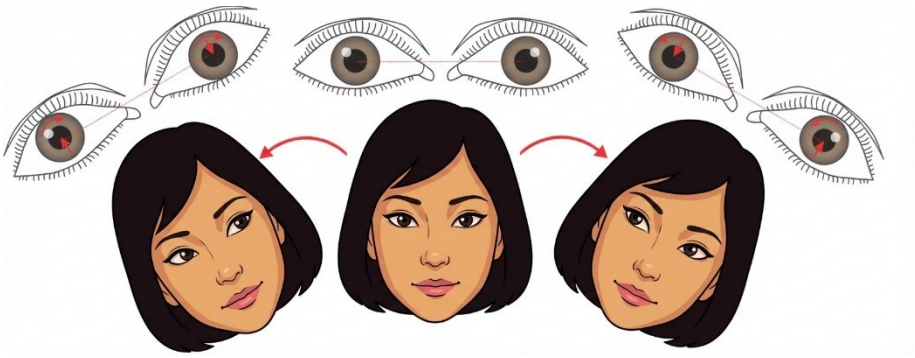
otolith organs. Indeed, the otolith organs are considered the oldest part of the vestibular apparatus, essential for detecting the ubiquitous force of gravity. Over the course of evolution, the ability to detect linear acceleration (including tilt via gravity) provided a survival advantage (e.g. for righting reflexes and spatial orientation), upon which the later-evolving canal system added specialized rotation sensing. This deep evolutionary conservation is reflected in the robust, direct pathways of the otolith reflexes, which like the VOR are organized for short-latency, life-sustaining responses.

Role of Otolith Organs in Normal Vestibular Function

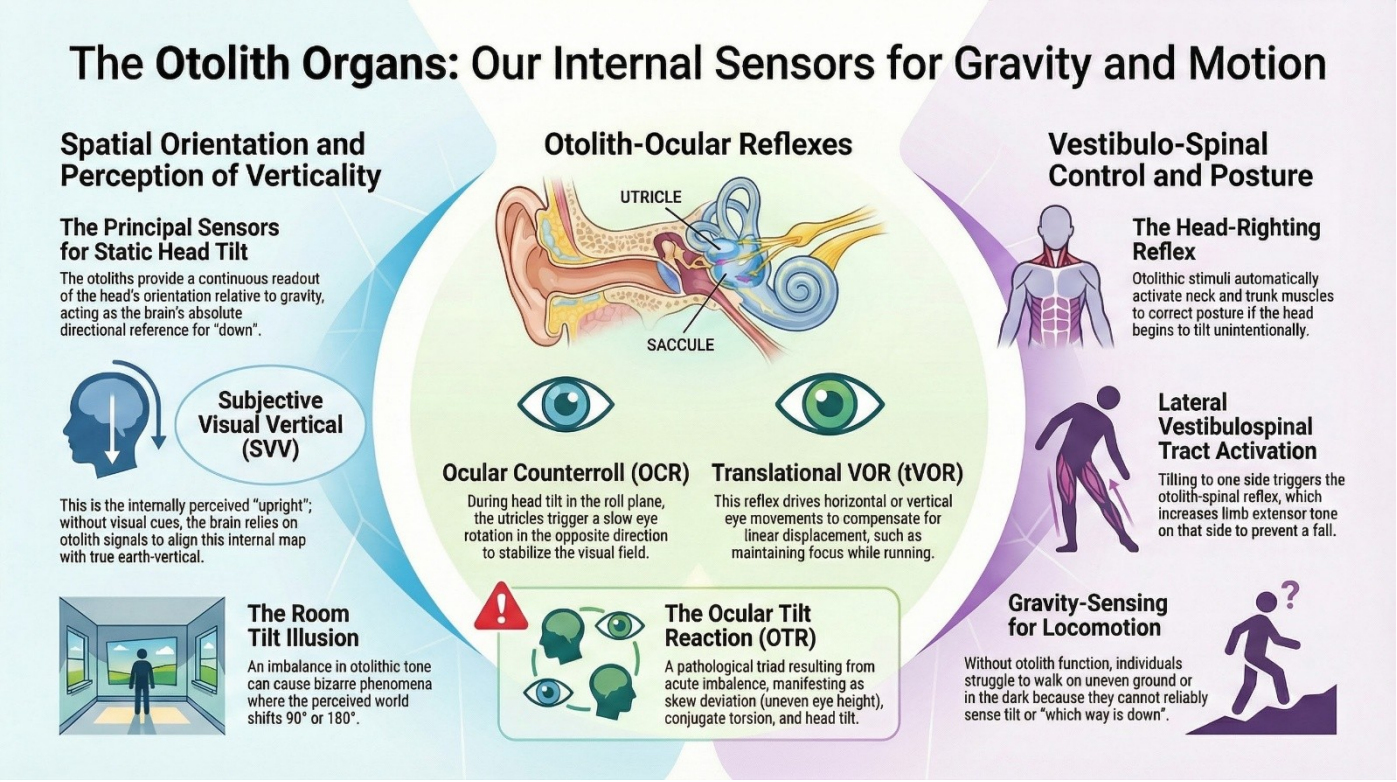
In normal physiology, the utricle and saccule serve three overarching functions: **orientation, ocular reflexes, and postural control.** Together, they encode our sense of head position in space and trigger compensatory eye and body movements that maintain equilibrium.

Spatial Orientation and Perception of Verticality: The otoliths are the principal sensors for static head tilt relative to gravity. They provide the brain with a continuous readout of the head's orientation with respect to the gravitational vector. This information is integrated centrally (in vestibular nuclei, thalamus, and cortex) with visual and proprioceptive cues to form our perception of verticality and spatial orientation. For instance, when you tilt your head to one side, the utricular afferents on that side decrease their firing while the opposite utricle's afferents increase, creating an imbalance that the brain interprets as head tilt. In response, your subjective visual vertical (the internally perceived "upright") tilts accordingly if visual references are absent. Normally, higher cortical processing (parieto-insular vestibular cortex, etc.) compensates so that you maintain a stable sense of the world, aligning your internal vertical with true earth-vertical. The otolith signals also contribute to cognitive maps of space and motion; without them, patients have distorted spatial orientation (e.g. feeling the world tilt or being pulled to one side). Notably, because gravity is ever-present, the otoliths provide an absolute directional reference (the gravitational down) which the brain uses to calibrate other senses. This is why an otolithic tone imbalance – whether from a peripheral lesion or a central pathway lesion – can cause profound tilts of perceived vertical and even bizarre phenomenology like the "room tilt illusion" (transient 90° or 180° shifts in perceived orientation).

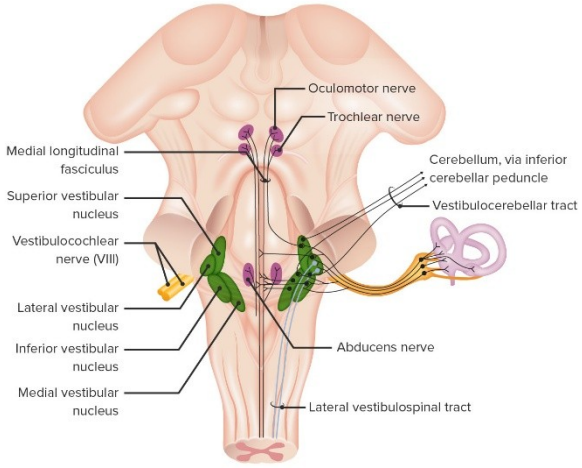
Otolith-Ocular Reflexes: A unique ocular motor contribution of the otoliths is the generation of compensatory eye movements for linear head motion and head tilts. During head tilt in the roll plane (ear downward toward the shoulder), the utricles mediate a slow torsional eye rotation in the opposite direction – a phenomenon called **ocular counterroll** – to help stabilize the visual field. For small head tilts, ocular counterroll is only on the order of a few degrees, but it minimizes retinal slip by partially righting the eyes toward earth-vertical. The **utriculo-ocular reflex pathways** underlying this involve projections from the utricular afferents to the vestibular nuclei and then to the ocular motor nuclei controlling the vertical rectus and oblique muscles, producing intorsion of the higher eye and extorsion of the lower eye [19]. In larger acute imbalances, this system can manifest as an **ocular tilt reaction (OTR)** – a pathological triad



of skew deviation (one eye down and the other up), conjugate ocular torsion, and head tilt, which will be discussed later. In translational movements (e.g. moving linearly forward/back or up/down), both utricle and saccule are activated and drive the **translational VOR (tVOR).** This reflex causes appropriate horizontal or vertical eye movements to compensate for linear head displacement, particularly important when viewing near targets. For example, if one steps to the right while fixating a near object, the eyes rotate leftward (and intort/extort appropriately) to maintain fixation. The tVOR is distance-dependent – requiring larger eye movements for nearer targets – and the otolith system works in concert with visual inputs to adjust the response. Although the translational VOR is less obvious clinically than the angular VOR, it is critical for maintaining stable vision during activities like running (where the head experiences repetitive vertical and fore-aft movements). In summary, the otolith organs contribute to eye movement control in the vertical and torsional planes that complements the canal-driven horizontal and vertical VOR. The integrity of these otolith-ocular reflexes is often tested via the manifestations of the ocular tilt reaction or with the subjective visual vertical, as discussed below.



Vestibulo-Spinal Control and Posture: The otoliths also drive reflexes that stabilize the body. Because they detect head tilt and linear acceleration, they trigger compensatory limb and trunk responses to maintain balance. A classic example is the **head-righting reflex:** if a person dozes off and their head starts to tilt, the otolithic stimulus from that tilt will reflexively activate neck and trunk muscles to correct posture. Likewise, tilting or accelerating to one side causes enhanced extensor tone in the limbs on that side via the lateral vestibulospinal tract **(the otolith-spinal reflex),** helping prevent falls to that side. The utricle in particular, being aligned with the plane of the head's base, is key for



sensing lateral tilts and sways, thus feeding into automatic postural adjustments. Patients with bilateral otolith loss may have relatively preserved canal-mediated VOR but still experience imbalance and lateropulsion because they lack graviceptive (gravity-sensing) inputs to guide their posture and perceive upright. In the context of locomotion and orientation, the otolith organs work continuously with proprioceptive and visual systems to ensure our centre of mass stays over our base of support and that we know "which way is down." Without otolith function, individuals have difficulty with tasks like walking in the dark on uneven ground, as they cannot reliably sense tilt or linear movement. Thus, normal vestibular function relies on the otoliths for gravity reference and linear motion feedback that underpin both conscious orientation and unconscious balance reflexes.

Clinical Assessment of Otolith Function

Bedside Evaluation: Otolith Reflexes and Perceptual Tests

Subjective Visual Vertical (SVV) and the bedside "Bucket Test" (static utricular graviception)

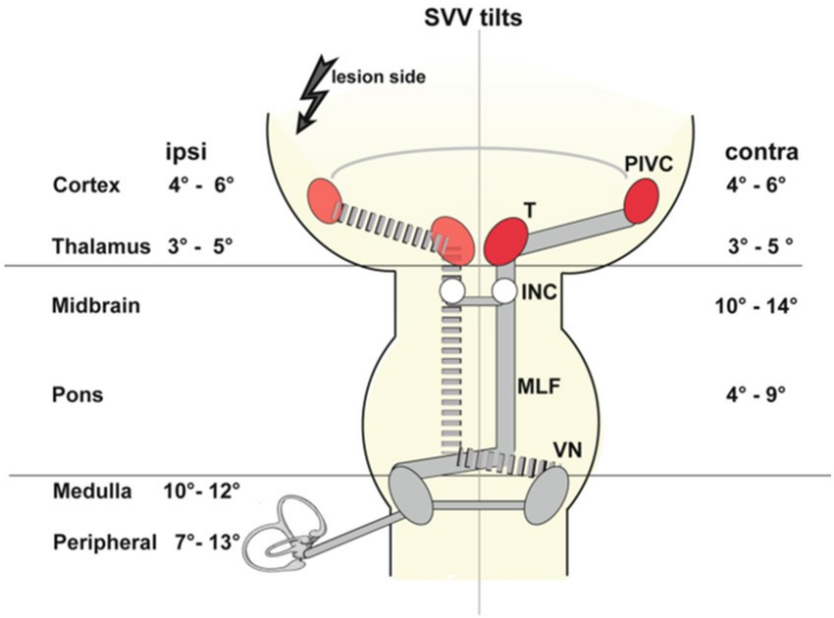
What SVV measures (the physiology you are interrogating) Subjective visual vertical (SVV) is a psychophysical readout of the brain's estimate of earth-vertical in the roll plane. Clinically, it functions as a high-yield marker of otolith (predominantly utricular) tone imbalance and its central "graviceptive" pathways. In routine vestibular clinics, otolith contributions are frequently inferred indirectly (e.g., skew/ocular tilt phenomena) or tested via VEMP paradigms rather than direct translational VOR quantification; SVV offers a pragmatic perceptual complement to these reflex-based measures.

VOR function testing

At the peripheral level, the utricle provides the dominant tonic reference for head roll-tilt and gravity-relative orientation, conveyed mainly via the superior vestibular nerve division (whereas the saccule is predominantly inferior division). This matters clinically because otolith tests can dissociate by nerve division (SVV/oVEMP patterns aligning with superior/utricular involvement; cVEMP aligning with inferior/saccular involvement).

VOR function testing

At the central level, SVV depends on multisensory integration, but lesion studies demonstrate a consistent anatomical logic: acute unilateral lesions along the graviceptive pathways produce SVV roll-tilt, and the direction of tilt follows the level of the lesion relative to pathway crossings in the brainstem. A large lesion–behaviour mapping study confirmed that ipsiversive SVV tilts are associated with pontomedullary structures including the medial vestibular nucleus and the MLF, whereas contraversive SVV tilts are



associated with more rostral pontomesencephalic structures including the rostral interstitial nucleus of the MLF, interstitial nucleus of Cajal, and oculomotor-related regions [22].

Conceptually, SVV is best treated as the perceptual correlate of roll-plane vestibular tone imbalance that also manifests motorically as **ocular tilt reaction components (skew deviation, ocular torsion, and head tilt).**

Why SVV is clinically valuable (and when it is higher yield than you expect)

SVV is particularly valuable when spontaneous nystagmus is absent/weak, when fixation suppresses peripheral nystagmus, or when the question is "is there a graviceptive pathway imbalance even if the canal tests are unremarkable?" In carefully phenotyped dizzy patients, **marked SVV deviation can be the only abnormal bedside sign of a small but clinically important dorsolateral medullary or pontine lesion**.

SVV also has a useful temporal profile: in acute unilateral vestibular dysfunction, SVV tilts often improve and normalize over weeks, tracking central compensation and recalibration. This makes SVV potentially useful as a longitudinal marker when symptoms persist, but objective canal measures have stabilized.

Core testing principle (the "rule" that governs all SVV methods)

All SVV methods must remove reliable visual orientation cues. If the patient can see a doorframe, examiner's face, monitor edges, or any stable frame, the test becomes a cognition-heavy "guess the room vertical" task and loses diagnostic yield.

A systematic review of SVV methods emphasized that protocols vary widely; nevertheless, for standardization and interpretability, SVV should be assessed without spatial cues (e.g., in darkness or with random-dot backgrounds) and repeated in an even number of trials (commonly 6–10) with the subject upright [21].

Normal SVV (mean adjustment error) is commonly considered to lie within approximately −2.5° to +2.5° under standardized conditions.

SVV paradigms (bedside vs laboratory)

Laboratory SVV systems typically use a hemispheric dome with random-dot backgrounds, computerized line adjustment on a dark field, or goggle-based SVV platforms. The dome method reduces external cues and is highly standardized; the computerized method is easier to deploy but can introduce subtle frame cues depending on screen borders and room conditions.

The bedside bucket test is a low-cost analogue method designed to reproduce the essential feature of the dome paradigm: a luminous/contrasting line presented in a cue-poor visual field while the examiner randomizes starting orientation.

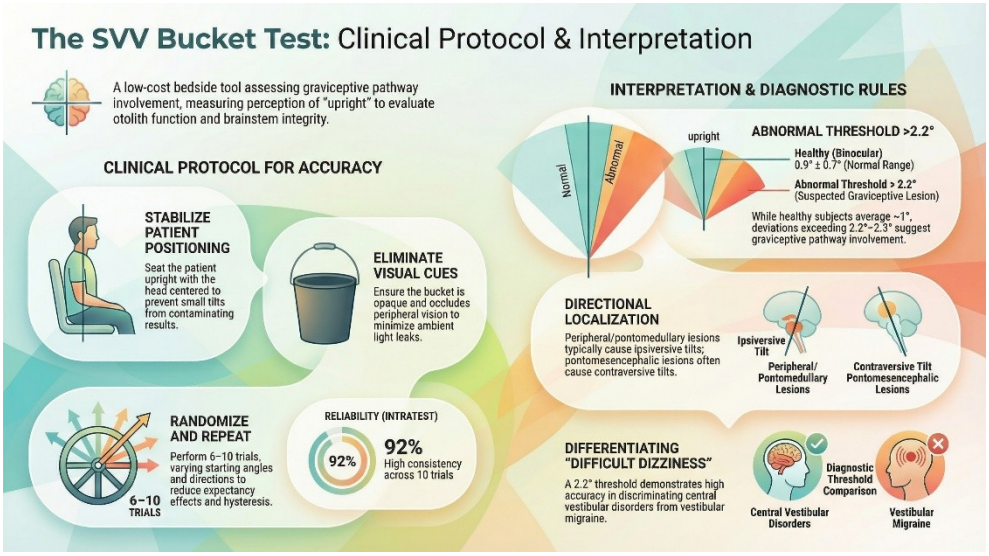
The bucket test: protocol (how to do it so the result is physiologically interpretable)

The validated "bucket method" uses a bucket with an interior diametric line and an exterior angular scale so the examiner can read the deviation at the moment, the patient declares the line is vertical. In one widely used clinical implementation, the bucket is opaque; the interior

bottom contains a straight line; the exterior bottom contains a protractor (or degree markings), and a weighted plumb line can be used as a reference for true vertical when reading the angle.

Key procedural features that determine interpretive quality:

- **Patient posture and head position:** seat the patient upright, with the head centred in the bucket rim. Even small head tilts can shift otolith tone and contaminate the estimate of "upright" verticality.
- **Removal of cues:** the bucket should occlude peripheral vision and minimize ambient light.
- **Randomized starting angle and direction:** begin each trial from a different roll offset and vary clockwise vs counterclockwise approaches to reduce hysteresis and expectancy effects.
- **Repeated trials and averaging:** at least 6 trials (often 6–10) improves reliability and aligns with standard recommendations in the SVV literature.
- **Record both signed and absolute deviation:** the signed error captures directionality (ipsiversive vs contraversive), while the absolute error captures magnitude.



In Zwergal's validation study, the bucket method showed high agreement with the hemispheric dome method and strong reliability: intertest reliability was ~89–90%, and intratest reliability across 10 repetitions was ~92%. Normative absolute deviations in healthy subjects were small (approximately 1° on average): 1.1 ± 0.7° (monocular) and 0.9 ± 0.7° (binocular) [9].

A practical abnormal threshold used in clinical studies is approximately 2.2–2.3° from true vertical (closely aligned with the commonly cited **±2.5° normative range**), with larger deviations strengthening suspicion for graviceptive pathway involvement.

Monocular vs binocular testing is not merely a technical variant. Monocular SVV can help when you suspect ocular alignment or cyclotorsional issues contributing to the percept, but

interpretation becomes more complex; in most vestibular bedside use-cases, binocular SVV is the practical default.

Interpretation framework: magnitude + direction + syndrome context

SVV should be interpreted as part of a roll-plane "tone imbalance package," alongside skew deviation testing, ocular torsion (if assessed), and head tilt posture. SVV tilts frequently co-occur with ocular tilt reaction components and tend to be directionally aligned in the roll plane.

Directionality can be diagnostically localizing, but only if you anchor it to the relevant neuroanatomical rule: because graviceptive pathways cross within the brainstem, peripheral or pontomedullary lesions typically produce ipsiversive SVV tilts, whereas pontomesencephalic lesions above the crossing more often produce contraversive tilts [22].

Magnitude also matters. In acute unilateral peripheral vestibular lesions (e.g., vestibular neuritis), SVV deviations can be marked early and then improve over weeks with compensation. In central lesions, deviations can be large and persist, but smaller tilts can also occur with thalamic or cortical lesions—sometimes without accompanying ocular torsion or skew deviation—reflecting a more "perceptual" verticality network disturbance rather than a classic full ocular tilt reaction.

A useful high-stakes bedside application is the "difficult dizziness" phenotype where vestibular migraine is a competing diagnosis. In one prospective study of patients with dizziness, **the SVV bucket test demonstrated excellent discrimination between MRI-confirmed central vestibular disorders and vestibular migraine (AUC ~0.9) using a threshold around 2.2°** [23]. This does not make SVV a standalone rule-out tool for stroke, but it can materially raise or lower suspicion when integrated with ocular motor findings and risk factors.

Indications (when SVV/bucket test is worth doing)

SVV and the bucket test are indicated when the diagnostic question includes: suspected utricular dysfunction; suspected graviceptive pathway involvement (brainstem/cerebellar); acute vestibular presentations where skew/roll-plane signs would materially shift urgency; chronic dizziness with unexplained postural bias or subjective tilt; and longitudinal follow-up where a compensation marker is desired. These indications are especially relevant when canal tests (vHIT/calorics/rotary) are normal or discordant, and a complementary otolith-centric measure is needed.

Contraindications and practical limitations

There are few true contraindications. The practical limitations are patient cooperation (cognition, attention), severe visual impairment that prevents line alignment, inability to sit safely upright, and inability to maintain head stillness. The method is also vulnerable to environmental cue contamination: any light leak or visible frame can normalize SVV despite a vestibular tone imbalance.

A subtle but important interpretive caution is device choice. Some "SVV" methods adopted from ophthalmology (e.g., Maddox-rod-like near-eye devices) can reflect the subjective perception of ocular cyclotorsion rather than environmental verticality, and therefore can misattribute an ocular motor disorder (e.g., extraocular muscle palsy) to vestibular dysfunction. This pitfall is avoided

by SVV paradigms that present a line in a cue-poor environmental field (dome, screen in darkness, or bucket test).

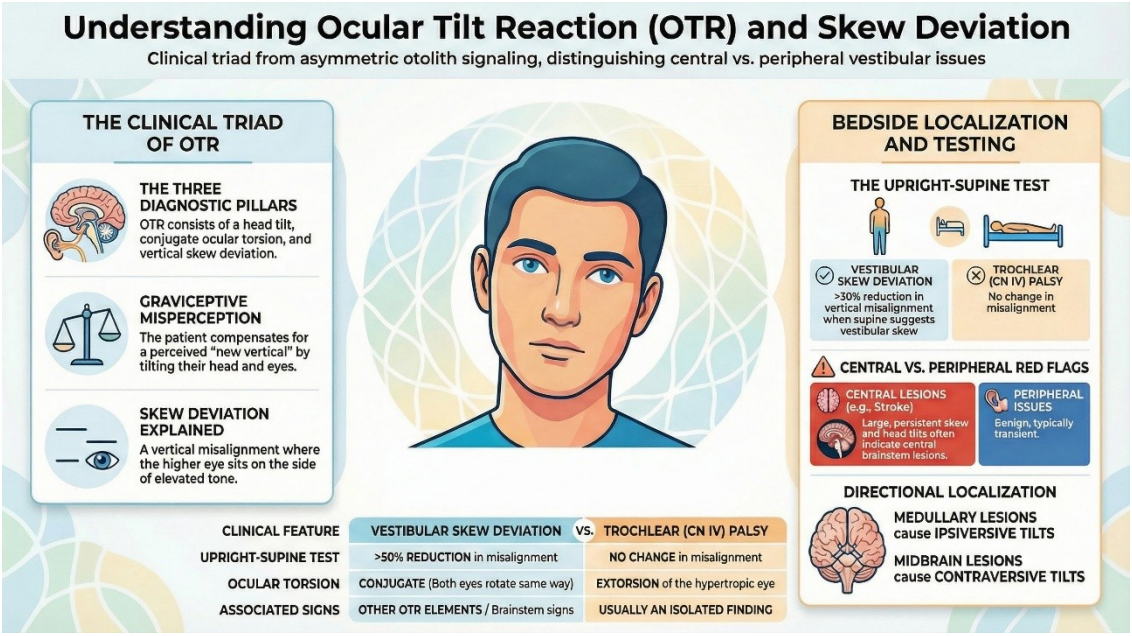
Pitfalls and common errors (the ones that create false reassurance or false alarms)

The most common source of false negatives is inadvertent spatial cues (bucket not opaque, room not dark, examiner visible). The most common source of false positives is head tilt within the bucket, particularly if the patient is guarding neck pain or has a habitual head posture. Overinterpreting small deviations near the cutoff is another common error; because normal subjects can have small biases, the magnitude, consistency across trials, and syndrome context should drive inference rather than a single trial.

Finally, SVV is not a screening test for “any vestibular disorder.” In a study of vestibular patients measured with the bucket test, ROC values for differentiating controls from vestibular patients were weak (<0.8), and no robust single cut-off separated groups well, supporting the concept that bucket SVV is best used as a targeted graviceptive/utricle-oriented probe rather than a broad screening tool [24].

Ocular Tilt Reaction and Skew Deviation: The ocular tilt reaction (OTR) is a clinical manifestation of asymmetric otolith (mainly utricular) signalling, whether from peripheral or central causes. It consists of a triad: (1) a **head tilt,** usually to the side of lower vestibular tone; (2) a **conjugate ocular torsion** (both eyes rotate toward the dependent ear); and (3) a **skew deviation** (a vertical misalignment of the eyes, with the higher eye on the side of the elevated otolith tone and the lower (hypotropic) eye on the side of reduced tone). In essence, the patient behaves as if gravity has been pulled off-center, so their eyes and head compensate toward the perceived new vertical [12].

Peripheral vs. Central OTR: A unilateral peripheral vestibular lesion (e.g. an acute right



labyrinthectomy) can produce an OTR with right head tilt, right eye intorted and down (hypotropic), left eye extorted and up – i.e. toward the lesioned side (ipsiversive OTR). This reflects the loss of right utricular input, causing the brainstem to treat the intact left utricle's activity as a head tilt to the left, and accordingly the eyes/head counter-roll rightward. Such peripheral OTR is often transient; as compensation occurs, the head tilt and skew deviation typically diminish over days, although subtle ocular torsion and SVV tilt may persist longer. In central lesions – classically lateral medullary infarction (Wallenberg syndrome) or dorsal pons infarcts – an OTR is a well-known sign, often more persistent. In Wallenberg syndrome, an ipsiversive OTR (head tilt and ocular torsion toward the lesion side) is common because the vestibular nuclei or their connections are disrupted. Midbrain lesions (e.g. affecting the interstitial nucleus of Cajal) can cause a contraversive OTR (head tilt opposite the lesion) due to disruption of decussated utriculo-ocular pathways [11]. Observing an OTR can thus be very localizing: an acute vestibular syndrome with a strong skew deviation and head tilt suggests a central lesion until proven otherwise, especially if accompanied by other brainstem signs. Conversely, a mild head tilt in isolation in a known peripheral disorder may be peripheral.

Skew Deviation Test (Cover Test of Skew): To detect a skew deviation, a simple bedside method is the alternate cover test: with the patient looking at a target, alternately cover each eye and observe the uncovered eye for a vertical corrective shift. A skew deviation is present if one eye consistently drifts up and the other down when uncovered, indicating a vertical misalignment due to otolith pathway imbalance [10]. In the context of the acute vestibular syndrome (the HINTS examination), a vertical skew deviation (especially if large and not obviously from a chronic trochlear palsy) strongly favours a central cause (stroke). It's important to distinguish skew from trochlear nerve palsy (CN IV palsy), which also causes a hypertropia and head tilt.

Upright-Supine Test: A useful clue is the positional change of the vertical deviation. In peripheral vestibular skew deviations, the vertical misalignment often lessens or disappears when the patient lies supine (gravity no longer accentuates the otolith imbalance), whereas in a fourth nerve palsy the hypertropia remains the same in supine position. The "upright-supine test" formalizes this: a >50% reduction in vertical misalignment when supine suggests a vestibular

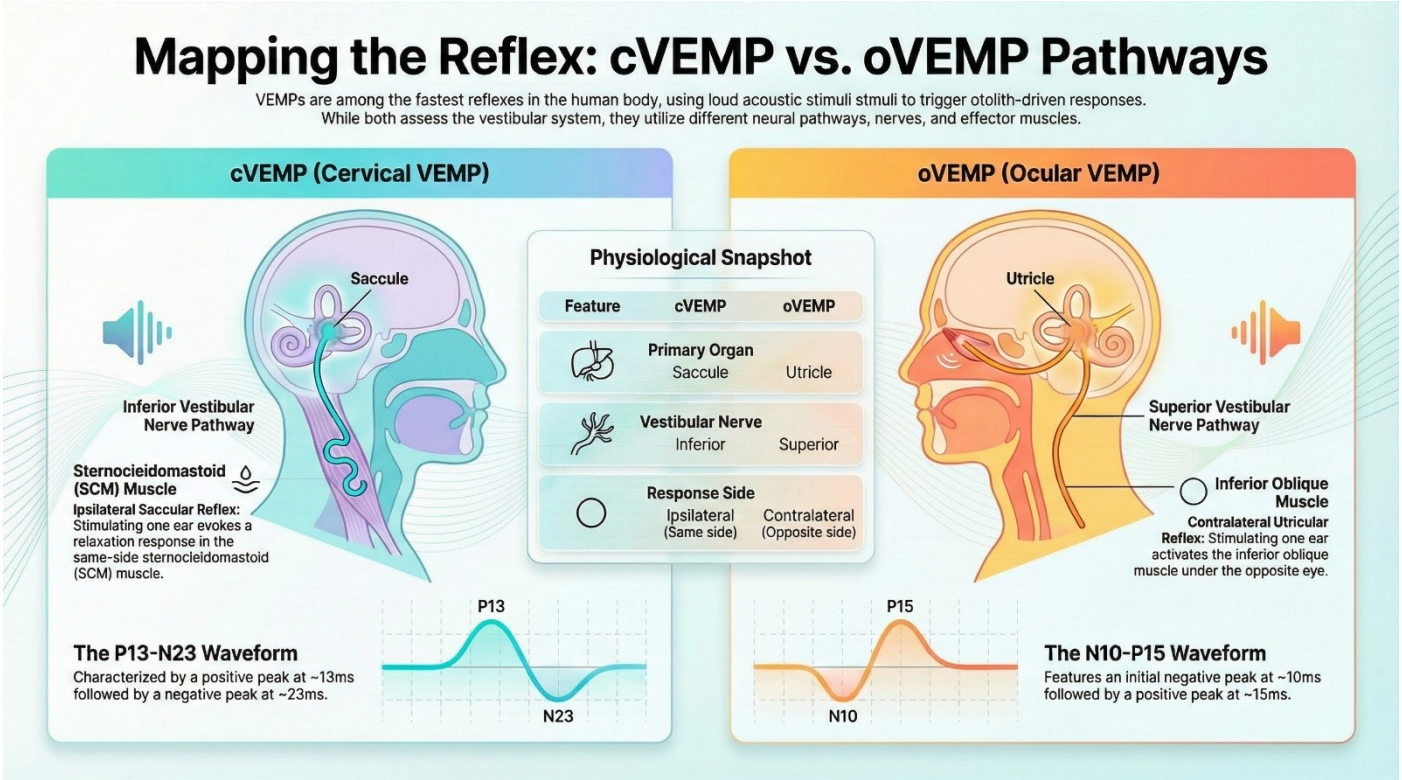
skew deviation (and thus warrants investigation for central lesion if not already explained), whereas no change suggests a trochlear palsy. Additionally, skew deviation usually comes with other OTR elements (torsion, head tilt, abnormal SVV) and other neurologic signs, whereas an isolated CN IV palsy will have torsion of the affected eye in the opposite direction (extorsion of the hypertropic eye) and a head tilt away from the hypertropic eye (opposite OTR pattern). Clinical relevance: Detecting an OTR or skew can be vision-saving or lifesaving. For example, in Wallenberg syndrome, an OTR often appears (sometimes the patient's first clue is that their world is tilted or they have torsional nystagmus); recognizing it prompts a search for lateral medullary signs. In superior canal dehiscence (a peripheral "third window" disorder), patients may show a subtle OTR or torsional nystagmus during loud sound or pressure stimuli (Tullio phenomenon), reflecting an otolith-canal crosstalk. And in acute vestibular neuritis, a small skew deviation might be transiently present – but if a large skew persists, one must reconsider a brainstem stroke. **Pitfalls:** When assessing skew, ensure the patient is fixing on a target (to control for fixation disparity). Small vertical phorias (latent misalignments) can mimic skew on alternate cover, but these usually have no torsion and are long-standing. Also be wary of baseline ocular misalignments: a chronic hypertropia from old strabismus is not a skew. The presence of conjunctival torsion (examined via fundus exam or noting torsion of iris landmarks) concurrent with skew is a telltale of OTR.

In summary, the OTR and skew deviation are key bedside signs of otolith pathway dysfunction – their detection and correct interpretation provide a direct window into vestibular tone imbalances in the graviceptive (gravity-sensing) pathways.

Laboratory Evaluation: Vestibular Evoked Myogenic Potentials (VEMP)

Among laboratory tests, vestibular evoked myogenic potentials have emerged as the most specific assays of otolith organ function. VEMPs are short latency electromyographic responses elicited by intense acoustic stimuli (sound bursts or bone vibrations) that activate the vestibular organs and produce reflex muscle contractions. There are two complementary variants: the **cervical VEMP (cVEMP),** recorded from the ipsilateral sternocleidomastoid (SCM) muscle, primarily reflecting saccular function and the inferior vestibular nerve; and the **ocular VEMP (oVEMP),** recorded from extraocular muscles (inferior oblique/inferior rectus) under the eyes, primarily reflecting utricular function and the superior vestibular nerve [7]. Together, cVEMP and oVEMP allow assessment of both otolith organs and their respective

nerve divisions.



Physiological Basis: A loud acoustic stimulus (air-conducted tone burst via headphones, typically 500 Hz, or a skull vibration) can excite vestibular afferents through bone conduction or fluid pathways. The saccule, despite being an organ of balance, has high sound sensitivity and can respond to loud sounds (in fact, the saccule may be evolutionarily related to auditory function in some species). The cVEMP pathway is a vestibulo-collic reflex: the saccular afferents (mainly carried in the inferior vestibular nerve) are activated by the stimulus, their signals travel to the ipsilateral vestibular nucleus, and via descending vestibulospinal projections (predominantly the MVST) influence the ipsilateral SCM motor neurons, causing an inhibitory postsynaptic potential that momentarily relaxes the contracting SCM [15]. The result is a brief EMG potential detectable as a biphasic waveform on surface electrodes over the SCM – **classically a positive peak around 13 ms (P13) followed by a negative peak around 23 ms (N23)**. This represents the first ~ biphasic response of the tensed muscle to the sound, and it is an ipsilateral response (stimulating the right ear evokes a response in the right SCM).

In contrast, the oVEMP pathway is a vestibulo-ocular reflex: utricular afferents (superior vestibular nerve) are stimulated and, through a polysynaptic route involving the vestibular nuclei and decussating pathways, lead to activation of the contralateral inferior oblique muscle (an extraocular elevator and excyclotorter). The oVEMP is recorded on the contralateral side, just below the eye (on the inferior orbital margin) while the patient looks upward to bias the inferior oblique. The oVEMP waveform is typically identified by an initial negative peak at ~10 ms (N10) followed by a positive peak ~15 ms (P15). Thus, a loud stimulus in one ear will elicit an oVEMP predominantly under the opposite eye. These latencies (~10–15 ms) make VEMPs among the fastest reflexes in the body – only a few synapses from ear to muscle – underscoring the primitive, direct nature of otolith reflexes.

Techniques and Parameters: Performing VEMPs requires specialized equipment (evoked potential system) and patient cooperation. For cVEMP, the patient is asked to elevate or rotate their head to activate the SCM (providing a tonic EMG background), and a series of loud click or tone-burst stimuli (e.g. 500 Hz tone bursts at 95 dB nHL, 5/sec rate) are delivered monaurally. Surface electrodes record the averaged waveform from the SCM. Key parameters measured are the P13 and N23 latencies (normally ~13 and ~23 ms in younger adults) and the amplitude of the P13–N23 response. Because amplitude can vary with level of muscle contraction and individual factors, the interaural amplitude difference is often used: an amplitude asymmetry ratio = |(Amp_left – Amp_right)| / (Amp_left + Amp_right)| ×100%. Typically, a ratio under about 35% is considered normal (upper limit ~34% in one large normative study).

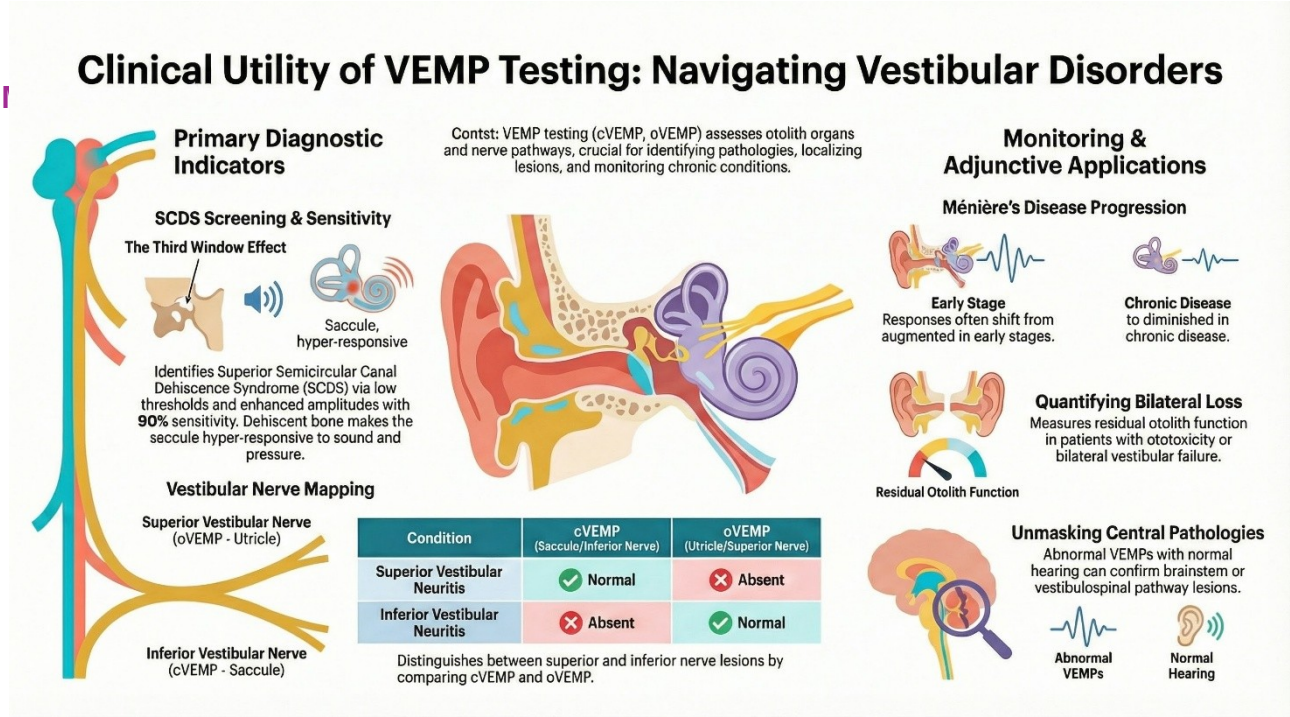
For oVEMP, the patient usually lies supine or sits and is instructed to gaze upward (30–40°) to bring the inferior oblique into activity. The same stimulus can be used (95 dB 500 Hz tone bursts), delivered via headphones or a bone oscillator on the forehead (e.g. midline at the hairline, known as Fz, for bone-conducted vibration which is very effective at eliciting oVEMPs). Recording electrodes placed infraorbitally (about 1–2 cm below the eye) captures the contralateral N10/P15 response. Because oVEMPs are smaller in amplitude than cVEMPs (often just a few microvolts), averaging many trials and minimizing background noise is important. Threshold can also be assessed: the lowest intensity that still yields a reproducible VEMP. In healthy young subjects, cVEMP and oVEMP are typically present up to at least 90–100 dB stimulation; the threshold is usually around 75–85 dB for cVEMP with air-conducted sound.

Indications and Clinical Utility: VEMP testing has become an invaluable tool especially for certain diagnoses:

- **Superior Canal Dehiscence Syndrome (SCDS):** Perhaps the clearest utility of VEMPs is in identifying this “third window” pathology. SCDS patients characteristically have low VEMP thresholds and enhanced amplitudes, reflecting the dehiscent bone over the canal that abnormally conducts sound pressure into the vestibule (making the saccule and utricle hyper-responsive). For example, a patient with SCDS may show clear cVEMP responses at 70 dB (where normal ears would have none) and oVEMP amplitudes several times larger than normal. In fact, the 2017 American Academy of Neurology guideline gave a Level C recommendation that cVEMP and oVEMP (threshold and amplitude) can be used to distinguish SCDS from normal ears, with good sensitivity/specificity (around 90% or higher) [7]. Thus, VEMP testing is a noninvasive screening that complements CT imaging in SCDS evaluation and can even lateralize the dehiscence by showing the enhanced responses on the affected side. Patients with SCDS often report Tullio phenomenon (sound-induced dizziness) and may have eye movements (e.g. torsional nystagmus) in response to loud sounds or pressure; the presence of robust VEMPs at low thresholds in these patients aligns with the pathophysiology of a pressure-sensitive “mobile window” in the bone.

- **Differentiating Vestibular Nerve Involvement:** Because cVEMP predominantly tests the saccule/inferior nerve and oVEMP tests utricle/superior nerve, combined VEMP results can help localize lesions in vestibular neuritis and other vestibulopathies. For instance, in superior vestibular neuritis (the most common type), one expects absent

oVEMP (utricle via superior nerve) and abnormal head impulse tests in the horizontal and anterior canals (also superior nerve), while cVEMP (saccule via inferior nerve) remains normal. Conversely, an inferior vestibular neuritis (rarer) might selectively abolish cVEMP (saccular response) with preserved horizontal canal function and oVEMP. In practice, an absent cVEMP in an acute vertigo patient with otherwise normal horizontal canal function suggests an inferior nerve lesion [6]. Vestibular schwannomas can also differentially affect divisions; an acoustic neuroma arising from the inferior vestibular nerve often leads to cVEMP loss as an early sign, whereas a superior vestibular nerve tumour might knock out oVEMP first. However, one must interpret these patterns with caution, as large lesions eventually affect both divisions, and there is individual anatomic variability.

- 

may show characteristic changes. In early or active Ménière's, some studies report augmented cVEMP amplitudes or lower thresholds (thought to reflect hydrops-induced increased pressure on the saccular macula), whereas in chronic or late-stage disease cVEMP responses may diminish or disappear (permanent saccular damage). Clinically, VEMP is not diagnostic of Ménière's by itself, but if a patient with unilateral hearing loss and episodic vertigo has a reduced cVEMP on the same side, it adds evidence of saccular hydrops. The 2017 AAN guideline, however, found insufficient evidence to say VEMPs definitively aid Ménière's diagnosis [7]. Still, some clinicians use serial cVEMP testing to monitor changes in otolith function in hydrops.

- **Central Vestibular Disorders:** VEMP testing is predominantly a peripheral test, but it can unmask central problems. A lesion in the vestibular nuclei or their descending pathways (e.g. lateral medullary infarct) might reduce or abolish the VEMP on the ipsilateral side, even if the ear is intact. For example, a patient with Wallenberg syndrome could have absent cVEMP due to damaged vestibulospinal pathways, mimicking an inferior vestibular neuritis on that side. Likewise, multiple sclerosis plaques in the brainstem have been associated with abnormal VEMPs in some cases (delayed or reduced responses) if the vestibular pathways are demyelinated. That said, no specific

central VEMP pattern is pathognomonic, so these tests are adjunctive. A normal VEMP does not rule out central vestibular dysfunction (since the lesion might spare the reflex or be more rostral in the pathway), but an abnormal VEMP with normal hearing in a brainstem stroke patient confirms involvement of the vestibular projections.

- **Bilateral Vestibulopathy:** In bilateral vestibular loss (e.g. ototoxicity from gentamicin), VEMPs are usually bilaterally absent or reduced, since both otolith organs are damaged. VEMP can thus help confirm the degree of bilateral vestibular hypofunction. It is one of several tests (along with head impulse, rotational chair, etc.) used to quantify residual function in each labyrinth [14]. Interestingly, some bilateral loss patients might have absent canal function but preserved otolith (or vice versa), so VEMP adds important data.

Contraindications and Safety: VEMP testing is noninvasive and generally very safe. The main "contraindications" are relative and relate to the stimulus and requisite manoeuvres. Patients with neck injuries, cervical spine instability, or recent neck surgery should not perform the head elevation needed for cVEMP, as the strain could be harmful. In such cases, the test can sometimes be done with the patient supine and lifting the head minimally (or not at all, using a slight shoulder lift to activate SCM) or avoided altogether. Similarly, severe back pain or orthopaedic issues may preclude the necessary positioning. Another consideration is that the sound stimuli for air-conducted VEMPs are loud (typically 95–100 dB HL); patients with active middle/inner ear pathology (e.g. a recent stapedectomy, tympanic membrane perforation, or profound hearing loss where loud sound could risk overstimulation) should be approached with caution. However, since we are stimulating vestibular cells, even deaf patients can have VEMPs (via bone conduction stimuli). Conductive hearing loss will attenuate or abolish air-conducted sound VEMPs by preventing the stimulus from reaching the saccule/utricle – this is not a contraindication per se, but a limitation (the test should then be done with bone conduction stimulation if possible). Overall, there are no absolute contraindications apart from inability to cooperate or inability to tolerate the stimulus. The acoustic stimuli are brief, and the risk of noise-induced cochlear damage is extremely low when using standard clinical protocols (the number of stimuli and intensity are within safe exposure limits). Still, patients with hyperacusis or tinnitus might find the test unpleasant. As a precaution, some clinics avoid VEMP in patients with recent ear surgery until healed, and perhaps in those with known perilymph fistula (to not provoke symptoms).

Results Interpretation and Caveats: A VEMP test is typically considered abnormal if: (a) the VEMP is absent or significantly reduced in amplitude on one side compared to the other (asymmetry ratio above the normal threshold, often >35–40%; (b) if latencies are markedly prolonged (this is less common except in demyelinating or brainstem conditions); or (c) if thresholds are significantly elevated (or responses only present at maximal stimulation) on one side compared to the other. When interpreting abnormal VEMPs, one must integrate other clinical information:

- **Absent cVEMP on one side:** If accompanied by hearing loss and vertigo, this could indicate labyrinthitis or vestibular nerve involvement (inferior division). If hearing is normal and head impulse on horizontal canal is normal, an isolated absent cVEMP could still be inferior vestibular neuritis or saccular loss. But check for conductive issues – even otitis media will abolish an air-conducted cVEMP. Tuning fork exams or an audiogram can help

ensure that an “absent cVEMP” isn’t due to a conductive block. Using a bone-conducted stimulus (like a 250 Hz skull tap or vibration) can bypass the middle ear; if that restores the VEMP, it implicates a conductive loss rather than saccular failure [13]. In older patients, note that cVEMP can be absent due to age-related saccular decline; normative data show a steep drop-off in amplitude beyond 60–70 years, and many healthy individuals over 70 have no recordable cVEMP [20]. Thus, absent VEMP in an elderly patient must be interpreted with caution and in context of other vestibular findings.

- **Absent oVEMP on one side:** This often suggests utricular or superior nerve dysfunction. If the patient also has an abnormal head impulse test (superior nerve) and perhaps superior canal paresis, it fits a superior vestibular neuritis or vestibular schwannoma on that side. But also consider that oVEMP can be technically harder to obtain; in some normal individuals (especially older), oVEMPs may be small or unrecordable. Ensure that the patient’s eyes were sufficiently elevated (to engage the inferior oblique) and that enough repetitions were averaged. Just as for cVEMP, a conductive hearing loss can reduce oVEMP (for air-conducted stimuli), but interestingly oVEMP tends to be easier to get with bone conduction vibration (which is not affected by middle ear) [16]. If using a tendon hammer tap to the forehead – a common technique for oVEMP – make sure patient safety (padding, etc.) and that the tap is delivered at consistent strength.
- **Reduced amplitude or delayed VEMP:** A small amplitude but present VEMP might still be within normal if symmetric. Delayed latency is uncommon in peripheral disorders (since it’s a quick reflex), so a noticeably prolonged P13 or N10 latency might hint at central conduction delay (e.g. multiple sclerosis in the vestibulospinal pathway) or simply a technical issue with stimulus timing. In bilateral vestibular loss, you may see bilaterally reduced amplitudes rather than outright absence, depending on extent of loss.
- **Hyperactive VEMP:** As noted, unusually large amplitudes and low thresholds point to disorders like SCDS or fenestrations (third window lesions). Also, some ototoxic medications can selectively affect canals more than otoliths, leading to an apparent “hyperfunction” of otolith reflexes relative to canals (though not truly pathologic hyperactivity, just preservation of otoliths).

Pitfalls in Technique: Perhaps the greatest source of error in VEMP interpretation is variability in muscle contraction. For cVEMP, if the patient does not contract the SCM equally on both sides, the raw amplitudes will differ. One should monitor EMG levels or use methods to normalize for background EMG (many systems now provide an “EMG normalization” feature). If a side has a very weak contraction, a reduced VEMP there might be falsely interpreted as abnormal. A practical approach is to repeat the test, coaching the patient to achieve similar effort on both sides, or to use rectified EMG averaging. For oVEMP, asymmetry in gaze angle or inability to maintain upgaze can affect results; fatigue of the extraocular muscle can also reduce amplitude. It’s important the patient keeps looking up and does not blink excessively during the averaging (blinks and eye movements create noise). Another pitfall: because oVEMP is a crossed response, a patient with a unilateral vestibular loss will show contralateral oVEMP loss. This sometimes confuses clinicians – e.g. a right-ear utricular failure gives a reduced oVEMP under left eye (since left eye response comes from right ear). To avoid mix-ups, always label which ear was stimulated for each oVEMP tracing. Finally, consider test-retest variability: VEMP amplitude can vary day to day and has a coefficient of variation that is not trivial. So, an asymmetry just at

the cusp of abnormal (say 38%) should be interpreted in light of the entire clinical picture and possibly rechecked rather than immediately ascribing pathology [8].

Emerging and Complementary Otolith Tests: Beyond the standard tests above, advanced vestibular laboratories may employ techniques like **off-vertical axis rotation (OVAR)** or unilateral centrifugation to isolate utricular function by creating continuous otolith stimulation. These are primarily research tools and not widely used clinically, so they are beyond our scope [18]. Likewise, dynamic SVV tests (measuring SVV during rotation or tilt) can unmask subtler otolith dysfunction. A mention should be made of galvanic vestibular stimulation (GVS) – delivering small currents behind the ear to activate vestibular afferents – which in research can test otolith vs canal contributions (otolith afferents are very sensitive to low-frequency galvanic stimulation). GVS is not a clinical test per se but has been used experimentally to assess otolith function where caloric and head impulses test mainly canals. For the practicing neuro-otologist, however, the **bedside ocular tilt tests and the VEMPs remain the main arsenal for evaluating otolith function.**

Conclusion

The utricle and saccule, with their elaborate otoconia-bearing maculae, form a foundational sensor system for gravity and linear motion. Their signals percolate through brainstem pathways to drive eye movements and postural reflexes and ascend to cortex to inform our sense of spatial orientation. In the dizzy patient, testing otolith function can uncover critical clues – a skew deviation pointing to a brainstem stroke, a tilted bucket test suggesting an acute unilateral vestibulopathy, or an absent VEMP localizing a vestibular nerve lesion. Modern vestibular practice has embraced VEMP testing as a valuable complement to canal-focused VOR tests, enabling a more complete evaluation of labyrinthine function [17]. As we have detailed, each otolith-specific test has its indications, nuances, and pitfalls: the SVV offers a quick window into graviceptive tone; the ocular tilt reaction links peripheral or central tonic imbalances to observable ocular signs; and VEMPs provide an objective, side-specific measure of saccular and utricular integrity. Matching the diagnostic rigor of gaze and VOR examinations, otolith assessments allow the clinician to achieve finely tuned lesion localization – distinguishing, for example, a superior nerve vestibular neuritis from a lateral medullary infarct or identifying the rare inferior nerve involvement. Ultimately, a thorough understanding of otolith organ function and testing equips the vestibular physician to “read” the often-subtle gravity-related signs in patients. In doing so, one can better restore patients’ equilibrium by targeting interventions (from rehab to surgical plugging of dehiscence) to the appropriate anatomic substrate. The careful integration of otolith findings with other vestibulo-ocular and neurological exam results exemplifies the art and science of neuro-otology – ensuring that no component of the balance system is overlooked in the quest to diagnose and treat vestibular disorders.

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Accuracy and Currency

While every effort has been made to ensure the accuracy and completeness of the content, vestibular medicine is a rapidly evolving field. Clinicians are encouraged to verify specific protocols, normative values, and therapeutic recommendations against current published guidelines and primary literature.

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All referenced works are cited in good faith for educational purposes. Where specific normative data or clinical criteria are cited, the original sources should be consulted for full methodological detail and applicability to individual patient populations.

Version History

Version 3.0 — April 2026 | Full ADC standard rebuild with front matter, table of contents, callout boxes, and image-preserving reformatting.

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